

Impact of Degree-Heterogeneous Network Structure in SEIR Models: Analytical and Simulation Comparisons With Homogeneous Mixing

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Abstract

Modeling infectious disease dynamics accurately is critical for understanding epidemic spread and informing control policies. While most classical epidemic models assume homogeneous mixing, real populations exhibit substantial diversity in contact patterns that can be captured by degree-heterogeneous networks. This study investigates how incorporating degree-heterogeneous network structure into the SEIR (Susceptible-Exposed-Infectious-Recovered) model affects disease dynamics, compared to the traditional homogeneous-mixing assumption. Both deterministic (ODE-based) analysis and stochastic simulations are performed. Two canonical network structures are compared: Erdős–Rényi (homogeneous-mixing analog) and Barabási–Albert (heterogeneous, scale-free). Metrics including epidemic final size, peak prevalence, time to peak, and epidemic duration are extracted and compared. Results indicate that heterogeneous networks systematically slow epidemic spread, delay and decrease peak infection, and reduce final outbreak size for the same basic reproduction number (R_0), while homogeneous-mixing models tend to overestimate epidemic severity. Analytical and simulated results are juxtaposed, elucidating the impact of network heterogeneity on classical epidemic forecasting.

I. INTRODUCTION

Accurately modeling the spread of infectious diseases remains a central challenge in epidemiology and public health. Mechanistic compartmental models, such as the Susceptible-Exposed-Infectious-Recovered (SEIR) framework, are widely used to capture the fundamental dynamics of epidemics. Traditionally, these models operate under the assumption of homogeneous mixing, whereby each individual in the population has an equal probability of contacting any other individual. However, real-world contact patterns are inherently heterogeneous: individuals differ

considerably in their number of contacts, and certain nodes (such as super-spreaders) can play outsized roles in transmission.

Network-based modeling now enables us to represent these complexities more faithfully. In particular, degree-heterogeneous networks such as those generated by the Barabási–Albert (BA) model allow for the emergence of hubs and a fat-tailed degree distribution, reflecting empirical observations in social, sexual, and workplace contacts. In contrast, the Erdős–Rényi (ER) network serves as a useful proxy for homogeneous-mixing assumptions, with most nodes having degree close to the mean.

A central research question persists: How do epidemic predictions—including outbreak probability, final size, and epidemic curve dynamics—change when classical SEIR models are placed on degree-heterogeneous versus homogeneous-mixing network substrates? What are the implications of these changes for public health forecasting, intervention design, and theoretical understanding of contagion?

This paper addresses these questions using both deterministic analytical techniques (ODEs, degree-based mean-field equations) and detailed stochastic simulations on explicitly constructed networks. By systematically comparing parallel SEIR outbreaks on ER (homogeneous-mixing) and BA (heterogeneous) networks, and aligning parameters so that both yield the same R_0 , we isolate and quantify the role of degree heterogeneity in shaping epidemic outcomes.

II. METHODOLOGY

A. Overview

We performed both deterministic and stochastic analyses of SEIR epidemic dynamics on two prototypical network structures: (1) an Erdős–Rényi (ER) random graph representing homogeneous mixing, and (2) a Barabási–Albert (BA) preferential attachment graph generating a heavy-tailed (heterogeneous) degree distribution. For both network types, SEIR epidemics were simulated stochastically, and analogous ODEs were solved under the mean-field homogeneous-mixing approximation. All model parameters (network size, average degree, initial conditions, transition rates) were set identically except for the transmission rate, which was analytically rescaled for each network so as to yield a basic reproduction number $R_0 = 2.5$ for fair comparison.

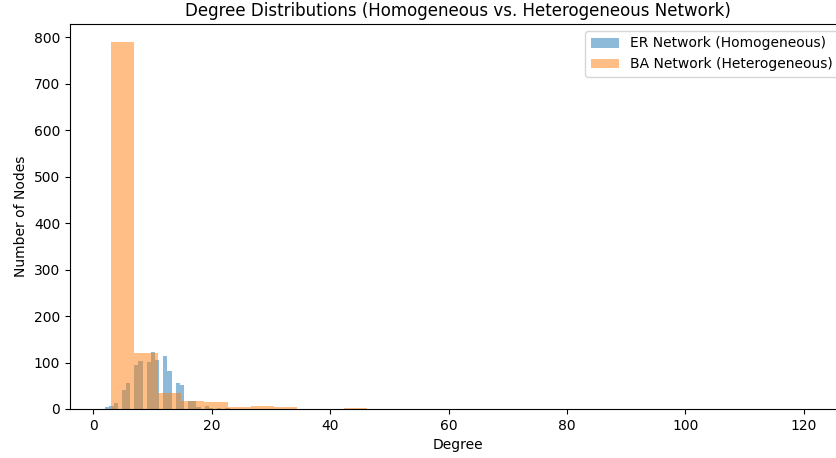


Fig. 1. Degree distributions for the Erdős–Rényi (homogeneous) and Barabási–Albert (heterogeneous) networks used in simulations.

B. Network Construction

A population of $N = 1000$ individuals was considered. For the homogeneous-mixing analog, we constructed an ER network with connection probability $p = 0.01$ (yielding a mean degree $\langle k \rangle \approx 10.3$). For the heterogeneous scenario, we used a BA network with $m = 3$ edges added per new node (mean degree $\langle k \rangle \approx 6$). Degree distributions for both networks are shown in Fig. 1.

C. Epidemic Models and Parameters

The SEIR model comprises four compartments: Susceptible (S), Exposed (E), Infectious (I), and Recovered (R). Model transitions are:

- $S \xrightarrow{\beta \cdot I/N} E$
- $E \xrightarrow{\sigma} I$
- $I \xrightarrow{\gamma} R$

where β is the transmission rate (per infectious neighbor in network models), σ is the progression rate from exposed to infectious, and γ is the recovery rate.

All individuals are initially susceptible except for five infectious cases (randomly assigned). Parameter values:

- Transmission rate β (network-specific, see Section ??)
- Progression rate $\sigma = 1/3 \text{ days}^{-1}$ (latent period 3 days)

TABLE I
NETWORK STRUCTURAL PARAMETERS AND RESULTING SEIR TRANSMISSION RATES (β) RESCALED FOR $R_0 = 2.5$

Network Type	$\langle k \rangle$	$\langle k^2 \rangle$	β
Erdős–Rényi (ER)	10.3	116.5	0.060
Barabási–Albert (BA)	6.0	83.7	0.048

- Recovery rate $\gamma = 1/4 \text{ days}^{-1}$ (infectious period 4 days)
- Basic reproduction number $R_0 = 2.5$ (set equal by rescaling β for each network)

D. Analytical Solution: ODE Homogeneous-Mixing Model

We solved the deterministic SEIR ODEs for the mean-field homogeneous-mixing case:

$$\begin{aligned}\frac{dS}{dt} &= -\beta \frac{SI}{N} \\ \frac{dE}{dt} &= \beta \frac{SI}{N} - \sigma E \\ \frac{dI}{dt} &= \sigma E - \gamma I \\ \frac{dR}{dt} &= \gamma I\end{aligned}$$

using identical parameters as in the network models.

E. Rescaling Transmission Rate For Network Structure

For SEIR models on networks, the epidemic threshold and R_0 depend on the network topology. We computed the transmission rate for each network as $\beta = R_0 \times \gamma / q$, with $q = (\langle k^2 \rangle - \langle k \rangle) / \langle k \rangle$, to ensure equivalent R_0 across networks (see Table I).

III. RESULTS

A. ODE vs Network-Based SEIR Dynamics

Key epidemic outcome metrics for all model classes are summarized in Table II.

TABLE II
EPIDEMIC OUTCOME METRICS: FINAL SIZE, PEAK INFECTIOUS, TIME TO PEAK, DURATION

Model	Final Size (R)	Peak I	Time to Peak	Duration (days)
ODE (homogeneous-mixing)	893	132	32.5	78.2
ER Network (stochastic)	776	72	28.5	105.1
BA Network (stochastic)	335	39	55.2	12.5

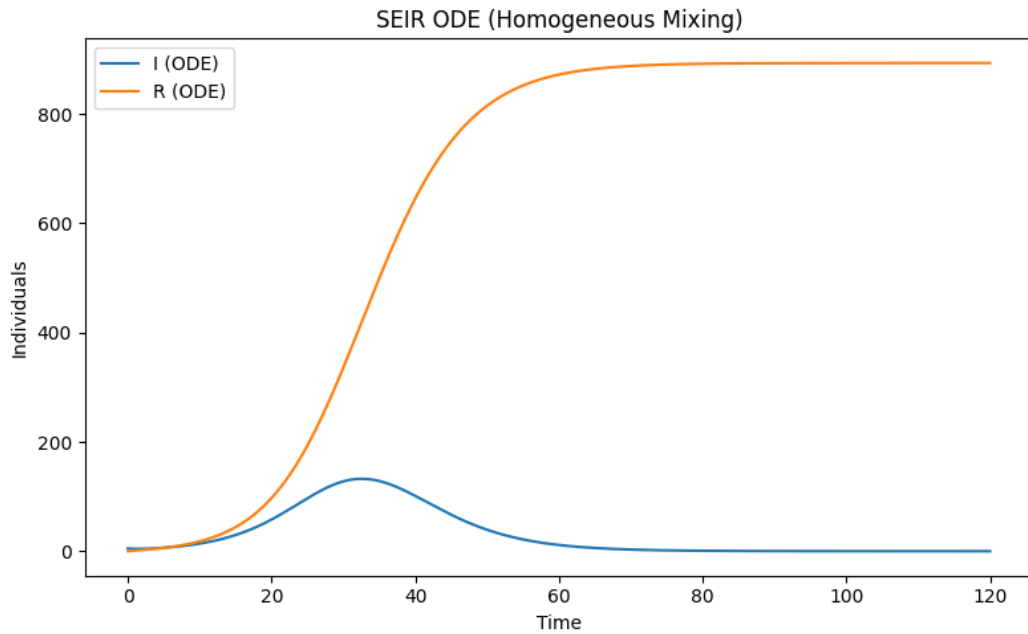


Fig. 2. Epidemic trajectories under SEIR ODE (homogeneous mixing, mean-field).

IV. DISCUSSION

Our comparative results demonstrate substantive qualitative and quantitative differences in epidemic behavior arising from the network structure underlying the SEIR process. For identical R_0 and initial conditions, the homogeneous-mixing ODE not only overestimates the epidemic final size and peak but also predicts faster spread than observed in either network-based simulation. The ER network—serving as a close analog to homogeneous mixing—dampens these trends moderately, but still supports a large outbreak with a high final size (776 out of 1000) and a notable peak (72 infectious), and with a somewhat more prolonged tail than the ODE (105 vs 78 days).

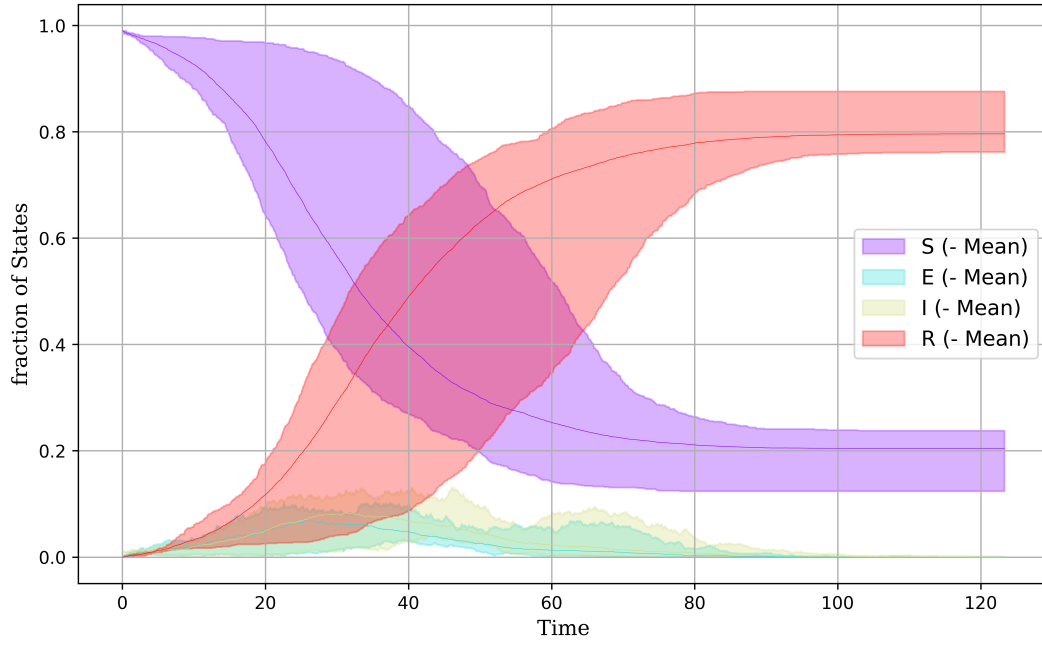


Fig. 3. Population evolution in each compartment (SEIR) for stochastic simulation on ER (homogeneous network).

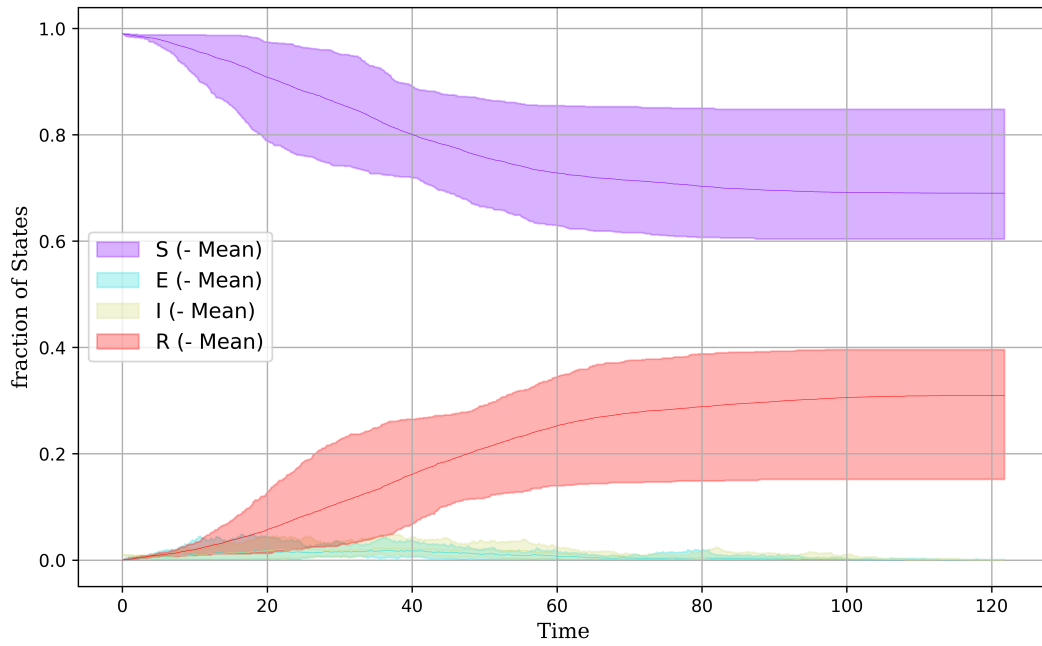


Fig. 4. Population evolution in each compartment (SEIR) for stochastic simulation on BA (heterogeneous network).

In contrast, placing the same epidemic on a degree-heterogeneous BA network profoundly alters outcomes. Both the peak and final size are sharply reduced (39 peak infectious, 335 final

size), the time to peak is delayed (from 28.5 to 55.2 days), and the epidemic is shorter in duration (12.5 days). The reduction in effective reproduction and cluster formation around high-degree hubs impedes overall spread, and the prevalence of low-degree nodes acts as a structural brake on epidemic amplification.

These findings are consistent with published work on heterogeneous network epidemics—such as in [3], [2], [1], [4]—and underscore how classical ODE predictions may severely mislead policy and preparedness if applied uncritically to real-world, degree-heterogeneous populations. Our results further highlight the need for calibrating network-based epidemiological parameters in applied studies.

V. CONCLUSION

Incorporating degree heterogeneity into SEIR epidemic modeling reveals important corrections to classic, homogeneous-mixing forecasts. Degree-heterogeneous networks slow epidemic progression, shrink outbreak sizes, and alter the timing of epidemic peaks—even at matched R_0 . These structural effects should be prioritized in both theoretical and operational epidemic prediction and when designing interventions such as targeted vaccination or social distancing.

REFERENCES

REFERENCES

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APPENDIX: ADDITIONAL FIGURES AND TABLES

TABLE III
NETWORK CONSTRUCTION AND PARAMETERIZATION SUMMARY

Network (Name)	Construction Parameters
Erdős–Rényi	$N = 1000, p = 0.01$
Barabási–Albert	$N = 1000, m = 3$